



Precipitating factors leading to decompensation in patient with chronic heart failure



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Background: Precipitating factors leading to exacerbation of chronic HF are often not directly related to the evolution of cardiac disease. The aim of this study was to examine prospectively the precipitating factors leading to hospitalization of patients with chronic HF.

Method: Potential risk factors that led to cardiac decompensation and subsequent hospitalization were studied in 346 patients between June 2013 to June 2015. Decompensation was defined as the worsening in the clinical NYHA class associated with the need for an increase in medical treatment (requiring at least iv diuretics).

Result: The mean age was 58 ± 14 years, 48% of patient were female and mean ejection fraction was 36%. One or more precipitating factors were identified in 84% of patients, nonadherence to diet (13%) or medications (22%) or to both (19%) was the most commonly identified precipitating factor (54%). Other precipitating factors were infections (16%), arrhythmias (17%), acute coronary ischemia (18.5%), and uncontrolled hypertension (2%). Miscellaneous causes were detected in 8% of cases (progression of renal disease 3%, anemia 2% and iatrogenic factors including usage of NSAIDs 3%).

Conclusion: Precipitating factor are frequently identified in patients hospitalized for HF. Many of these risk factors are avoidable and their identification is important in optimizing the management of HF. Patient noncompliance with prescribed medical regime accounts for more than half of the HF decompensation in this study. Thus patient education is very important to improve compliance and thereby minimize recurrent hospital admissions.

Reversible doxorubicin cardiomyopathy: A case report



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A middle aged male patient diagnosed with B-cell non-Hodgkin's lymphoma and was treated with rituximab, doxorubicin, vincristine, and cyclophosphamide (R-CHOP) chemotherapy

regimen for 6 months. Four months after the chemotherapy, patient presented with symptoms of heart failure. There was no significant history of any cardiac disease in the past. Chest X-ray was done which showed enlarged cardiac silhouette. Echocardiography was done which revealed a dilated cardiomyopathy with severe left ventricular (LV) systolic dysfunction. Chest X-ray before starting the chemotherapy had normal cardiac silhouette. Echocardiography reports before starting the chemotherapy were with normal limits with normal sized heart and normal ejection fraction. Patient was managed with optimal drugs and echocardiography was repeated after 1 year which showed an improved LV systolic function with ejection fraction of around 40%. This led to the diagnosis of doxorubicin-induced cardiomyopathy.

Cardiac cachexia



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Introduction: Heart failure is currently appreciated as a systemic and other multi-organ syndrome. The myocardium, peripheral tissues and organs are affected by metabolic failure, resulting in a global imbalance between catabolic and anabolic signals, leading to tissue wasting and, ultimately, to cachexia.

Cachexia is recognized today as severe complication of CHF that worsens clinical symptoms and carries a particularly grave prognosis. Mortality in HF patients with cachexia was as high as 50% at 18 months of follow-up, compared with 17% in cachectic patients (48).

Pathophysiology:

- The neuroendocrine activation – it is the cornerstone of current pathophysiological understanding of heart failure
- Inflammatory activation
- Metabolic impairment – metabolic regulation is increasingly recognized as contributing both to major symptoms (muscle weakness, fatigue, exercise limitation, and dyspnea) and to disease progression

Anabolic failure: The normal anabolic response to insulin and amino acid stimulation was reduced in HF patients by more than 50% because of both a blunted protein anabolic response and increased proteolysis.

Catabolic activation: Catabolic activation is particularly apparent in adipose tissue, as several independent pathways exert lipolytic signals on hormone-sensitive lipase.

Insulin resistance: The normal anabolic response to insulin and amino acid stimulation was reduced in HF patient by more than 50%, because of both a blunted protein anabolic response and increased proteolysis. Even in nondiabetic patients, insulin resistance in HF progresses in parallel to HF severity and predicts impaired functional capacity of cardiovascular and particularly of muscle function.

Skeletal muscle: Skeletal muscle is the main effector organ for physical activity and the body's largest amino acid storage pool.

Summary:

- Heart failure
- Metabolic failure
- Catabolic dominance signals
- Anabolic blunted signals
- Tissue wasting
- Cardiac cachexia